inflammatory drugs were taken and she began a course of desensitisation to wasp venom.

Comment

Immunity to bee stings is well known among beekeepers, indeed it appears that people get temporary relief from the pain of arthritis if they sustain several bee stings. Allergy to insect stings is a local or system type I immediate hypersensitivity reaction mediated by IgE. The effect of allergy to anti-inflammatory drugs may not be the same in all subjects, as has been shown in asthmatic patients sensitive to aspirin.[2] Treatment with anti-inflammatory drugs is widespread and may be overlooked in victims of bee stings as a possible complicating factor.

We have found no previous report saying that diclofenac, ibuprofen, or other non-steroidal anti-inflammatory drugs can reversibly modify the immune response. Though changes in specific tests have been shown in animal and in vitro experiments,[3-5] no noticeable general immunosupression has been reported in a clinical situation.

We think that all beekeepers should be warned of this possible hazard if prescribed non-steroidal anti-inflammatory drugs.

1 Ewan PW. Allergy to insect stings: a review. *J R Soc Med* 1985;78:234-9.
2 Asad SL, Kemeny DM, Youlten LJF, Frankland AW, Lessof MH. Effect of aspirin in "aspirin sensitive" patients. *Br Med J* 1984;288:745-8.
3 Aleksandrowa GM, Roshkowa WN, Borissowa LN. Study comparing immunotrophic effects of diclofenac sodium, indomethacin and sodium salicylate. *Farmakol Toksikol* 1981;44:450-3.
4 Lang G, Phumb N, Kraus H. *Influence of diclofenac on PHA-lymphocyte: stimulation in RA patients and controls.* Bethesda, Maryland: International League against Rheumatism, 1977:256. (Abstracts from XIV International congress of rheumatology.)
5 Wildfeuer A. Effect of anti-rheumatic drugs on the release of lysosomal enzymes from human leukocytes. *Z Rheumatol* 1984;43:23-6.

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Social class and age distribution in Reye's syndrome

We carried out a study to see whether social class affects the age of onset in fatal cases of Reye's syndrome in Great Britain.

Patients, methods, and results

The age at death and the father's occupation (obtained from the death certificate) in all 72 officially recorded fatal cases of Reye's syndrome that occurred in the British Isles between August 1981 and April 1984 were obtained from the joint surveillance scheme for Reye's syndrome of the British Paediatric Association and the Communicable Disease Surveillance Centre. The fathers' occupations were classified, blind to age, into six social groups (1, professional; 2, intermediate; 3, skilled non-manual; 4, skilled manual; 5, partly skilled; 6, unskilled) using a modification of the Registrar General's scheme (table). Seven of the 72 were unclassifiable. There was a significant negative correlation (Spearman correlation coefficient $-0{\cdot}412$, $t=-3{\cdot}5889$, $p=0{\cdot}0005$) between ranked social class and ranked age at death in months. There was also a significant difference between the observed number of cases in each social class and the number expected from the distribution of social classes in the general population ($\chi^2=47{\cdot}72$, $p<10^{-6}$).

Comment

The joint surveillance scheme has attempted to register all cases of Reye's syndrome occurring in the British Isles since 1981.[1] Data from death certificates are included and therefore under-registration is probably less likely with fatal than with non-fatal cases. Underdiagnosis, however, could be a considerable problem with this rare condition, as could the inclusion of certain inborn errors of metabolism that are indistinguishable from Reye's syndrome unless detailed metabolic studies are carried out.[2] Furthermore, no precise or universally agreed determinant of social class or social conditions exists, and classification is further limited when only self descriptions of fathers' occupations are available.

There are many possible differences between social groups, including geographical location, diet, exposure to environmental toxins, genetic constitution, and access to medical facilities. These could all affect the incidence, outcome, or diagnosis of Reye's syndrome, but why they should affect the age of onset is unknown. One possibility is that Reye's syndrome comprises at least two diseases, one of which is associated with social disadvantage and occurs early while the other is associated with social advantage and occurs later in life. The observed excess of cases in social groups 1 and 6 provides some support for this view (table).

Another possible explanation arises from the fact that diseases caused by common micro-organisms spread by the faecal-oral route or close personal contact show this pattern of age at onset. The age incidence of first contact with a common organism falls rapidly through childhood, in a manner analogous to half life decay. If improved social conditions cause the organism to circulate more slowly then the incidence of first contact falls more slowly and the mean age at onset rises.

Age at death of children with Reye's syndrome by social class

	Social class						
	1	2	3	4	5	6	All
Age at death (months):							
Mean age	106	55	39	55	28	20	50
Median age	118	28	16	51	9	6	25
Interquartile range	40-165	16-85	10-59	5-77	6-45	4-19	6-77
Age range	27-180	1-170	5-112	1-146	1-85	0-95	0-180
No (%) of cases	8 (12·3)	17 (26·2)	7 (10·8)	13 (20)	7 (10·8)	13 (20)	65 (100)*
% Of people in general population in each social class†	6·6	25·1	10·1	39·7	14·2	4·3	100

*Seven missing cases.
†Social class of economically active or retired husbands of married couples aged 16-44 in private households with one or more dependent children, England and Wales, 1981 census, 10% sample. Calculated from Household and Family Composition (CEN 81 HFC) table 28.

Reye's syndrome often occurs after viral infections of the respiratory tract,[2] though many of these viruses, such as influenza B, cause disease throughout life and are not confined to the age group in which Reye's syndrome occurs.[3] Viruses can, however, disturb the bacterial flora in the respiratory tract and lead to overgrowth of common Gram negative bacilli.[4] Furthermore, although there is no evidence that viraemia or bacteraemia directly causes Reye's syndrome, the disease has a clinical picture of toxaemia,[2] and animal models of the disease have been developed using bacterial toxins as causative agents.[5] Thus one possible explanation for our findings is that in some cases Reye's syndrome occurs because a chance first encounter with common toxigenic bacteria after a viral infection leads to bacterial overgrowth and toxaemia before immunity has developed.

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1 Hall SM, Bellman MH. British Reye's syndrome surveillance scheme, methodology—results. In: Pollack JD, ed. *Reye's syndrome IV*. Bryan, Ohio: National Reye's Syndrome Foundation, 1985:32-47.
2 Mowat AP. Reye's syndrome: 20 years on. *Br Med J* 1983;286:1999-2000.
3 Sullivan-Bolyai JZ, Corey L. Epidemiology of Reye's syndrome. *Epidemiol Rev* 1981;3:1-26.
4 Ramfrez-Ronda CH, Fuxench-Lopez Z, Nevarez M. Increased pharyngeal bacterial colonization during viral illness. *Arch Intern Med* 1981;141:1599-603.
5 Yoder MC, Egler JM, Yudkoff M, Chatten J, Douglas SD, Polin RA. Metabolic and mitochondrial morphological changes that mimic Reye's syndrome after endotoxin administration to rats. *Infect Immun* 1985;47:329-31.

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